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Family Practice Recertification

Decompression sickness is a complex condition that can present a considerable challenge to the health care provider. The mainstay of treatment for decompression sickness is therapeutic recompression therapy while breathing oxygen. The patient should be compressed as soon as possible, however patients should be considered for recompression even after several days delay. Aggressive hydration and high flow oxygen shuld be instigated, even before starting recompression therapy. The use of hyperbaric oxygen is generally safe, relatively non-toxic at clinical treatment depths, and can be used to treat young children. Pharmacologic agents may be useful adjuncts to recompression therapy but their proper role requires further study. Early consultation with a physician trained in dive medicine should be sought.

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Decompression Sickness, Evaluation and Treatment

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Confusion, headache, and joint pain after scuba diving

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Patient presentation

T.W., a 27-year-old white woman, came to the family medicine clinic complaining of a severe, generalized headache and fatigue that had persisted for 14 hours. For the last 12 hours, she had also suffered from progressive right shoulder pain, bilateral wrist and ankle pain, and tingling in the feet. She had no history of any significant medical problems or recent acute illness or trauma.

By the patient's own account, she had been scuba diving 18 hours earlier. While her first dive of the day—a 40-minute descent to a depth of some 36 feet of sea water (fsw)—was uneventful, her second dive had ended abruptly. During this 15-minute descent to 40 fsw, she was unable to clear her face mask of water; panicking, she rapidly ascended to the surface.

Only 8 hours before arriving at the clinic, the patient had visited the emergency department of a local hospital where her symptoms were diagnosed as musculoskeletal strain and fatigue. At that time, she was given a prescription for ibuprofen (Ibu-Tab, Motrin, Rufen, etc.) in 800-mg doses to be taken three times daily. She was also taking norgestrel and ethinyl estradiol (Lo/Ovral) once daily on a regular basis.

Diagnosis

Physical examination revealed a slightly confused, slender, young woman with poor concentration. Her vital signs were temperature, 98.2°F; blood pressure, 106/74 mm Hg; pulse, 78 beats per minute; respirations, 24 per minute. Laboratory results showed a complete blood cell count and electrolyte levels in normal ranges. Pertinent physical findings included poorly localized, deep, throbbing pain, 9/10 in intensity in the joint area of the right shoulder and in both ankle joints; this pain did not worsen with movement. Involved areas were not erythematous, swollen, or tender to the touch. Strength in her right shoulder was decreased compared with that in her left, and the strength of her ankle flexion and extension was decreased bilaterally. Diffuse paresthesia occurred in both feet and extended to the ankles.

Neurologic findings were significant; the patient was well-oriented but appeared to have problems concentrating, demonstrated by her inability to recite serial multiples of seven or repeat simple phrases. Her illness was diagnosed as decompression sickness (DCS), and she was immediately started on 100% oxygen by anesthesia mask. Vigorous hydration with Ringer's lactate was also begun intravenously. She received 325 mg of aspirin orally and was quickly transported to a nearby recompression, or hyperbaric, chamber for definitive treatment. There she received hyperbaric oxygen treatment (US Air Force Table 6), and her symptoms resolved. She was observed overnight in the hospital and discharged the next day after a careful examination showed no residual neurologic or other abnormalities. She was told not to dive or fly for at least 48 hours.

Discussion

DCS, most frequently associated with compressed gas diving, is a medical emergency. In 1992, the illness was treated in approximately 600 patients in North America.[1,2] With an estimated 2-3 million trained recreational scuba divers in the United States and about 500,000 new divers starting training each year,[3] it is becoming increasingly important for primary care physicians to be able to

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References: 1. Study 21097, data on file, 3M Pharmaceuticals. 2. McFadden ER Jr, Gilbert IA. Medical progress: asthma. *N Engl J Med* 1992;327:1928-1937. 3. Shim C, Williams MH. The adequacy of inhalation of aerosol from canister nebulizers. *Am J Med* 1980;69(6):891-894.

recognize, evaluate, and treat patients with DCS.

This illness is induced by inert gas bubbles, usually nitrogen, that have formed in the vascular system and tissues in enough volume to interfere with or alter organ or neurologic function. Diving while using compressed air may produce these bubbles. As a diver descends, the partial pressures of oxygen (P_{O_2}) and nitrogen (P_{N_2}) increase in the alveoli. A new tissue equilibrium is established, with higher levels of these gases dissolving in solution (Henry's law). During ascent, partial pressures decrease and the gases start to diffuse from the tissues. Although oxygen is removed through metabolic consumption, diffusion, and blood flow, inert nitrogen can be removed only by the latter two routes. If the rate of ascent exceeds the rate at which the inert gas can be eliminated from the body tissues, nitrogen bubbles may form, causing local symptoms due to vascular occlusion, local tissue distortion or disruption, hypoxia, or activation of the complement system and coagulation cascades.

Clinical manifestations of DCS range from skin eruptions and joint pain to neurologic dysfunction, profound fatigue, and weakness. Cardiovascular shock and death result in rare cases. Symptoms usually occur within 24 hours of a dive and can be progressive if untreated. Symptom onset 36 hours or more after a dive is uncommon. Untreated DCS may lead to significant functional or neurologic disability. Any neurologic finding, especially if progressive, usually signifies increasing severity of illness. Other structures, including the vestibular apparatus and lymphatic system, may also be affected.

Although the illness is more commonly associated with dives deeper than 33 fsw, there is considerable individual variability, and symptoms have been observed after shallower dives.[4,5] Regardless of the circumstances, time between symptom onset and treatment must be minimized.

Management

For nearly 100 years, the standard preliminary therapy for DCS has been 100% oxygen.[6] The patient should be placed in a supine position, and the oxygen—in a concentration as close to 100% as possible—

administered continuously through a tightly sealed oral-nasal mask until definitive treatment is started. The 100% oxygen increases the concentration gradient for the diffusion of nitrogen from bubbles back into the tissues and blood and then to the lungs where it is expelled.

Nitrogen bubbles may cause platelets and leukocytes to aggregate, leading to microvascular impairment and occlusion. Damage to capillary endothelium leads to increased vascular permeability, and some patients therefore show an increased hematocrit and platelet count. In severe cases of DCS, which are associated with a generalized capillary leak syndrome and hemoconcentration with increased blood viscosity, aggressive hydration is very important. Oral or intravenous hydration allows the continued washout of nitrogen from the tissues; at the same time, the process dilutes platelet aggregation and improves vascular flow. Isotonic crystaloid solutions, such as normal saline or lactated Ringer's, are best; glucose solutions are usually avoided. The use of corticosteroids, anticoagulants, and lidocaine HCl is controversial.[4]

If treated soon after the onset of symptoms, less severe cases of DCS may be resolved at this point in therapy. Before treatment is ended, however, consultation is warranted with a physician knowledgeable about recompression therapy, or hyperbaric medicine, the only known definitive treatment for DCS.[7]

Modern recompression therapy almost always employs oxygen-enriched breathing mixtures, or hyperbaric oxygen. When inhaled under pressure, oxygen dissolves in the plasma. At 3 atmospheres of absolute pressure (ATA), or 66 fsw, an arterial oxygen pressure of more than 2,000 mm Hg may be forced into plasma solution. By Boyle's law, recompression therapy also greatly reduces the size of nitrogen bubbles. The simultaneous use of 100% oxygen significantly increases the oxygen gradient, causing the inert nitrogen to diffuse from the bubbles back into the tissues, thus accelerating the rate of bubble nitrogen elimination.

Among its other benefits, therapy with hyperbaric oxygen offers greater oxygen delivery to support ischemic tissue and inhibits the further uptake of inert nitrogen[6]; the vasoconstrictive effect of oxygen may also reduce edema in some injured tissues. The optimum pressure for treatment is not known. Although most patients are successfully treated with a single oxygen recompression table to a depth of between 2 ATA and 2.8 ATA, individual responses may vary. Some patients may require repetitive hyperbaric oxygen therapy until symptoms are resolved or incremental benefit ceases.[4]

Conclusion

In treating DCS, recompression with 100% oxygen should begin as soon as possible and be considered even after several days' delay.[4] Aggressive hydration and high-flow oxygen should be initiated immediately. Hyperbaric oxygen is generally safe and relatively nontoxic at clinical treatment depths and can be used in children. Pharmacologic agents may be useful adjuncts, but their role is undefined. A physician trained in dive medicine should be consulted early.

More information on DCS and its treatment, including a list of hyperbaric treatment facilities in your area, is available from the Divers Alert Network, 919-684-2948, or the Undersea and Hyperbaric Medical Society, 301-530-9275. The Divers Alert Network emergency hot line is 919-684-8111. §

Opinions, interpretations, conclusions, and recommendations expressed in this article are the author's and are not necessarily endorsed by the US Air Force.

REFERENCES

1. Bove AA, Davis JC. Diving medicine. Philadelphia: WB Saunders Co, 1990:170-187.
2. Davis JC. Medical examination of sport scuba divers. San Antonio, Tex: Medical Seminars Inc, 1986:1-45.
3. Divers Alert Network. Report on diving accidents, Divers Alert Network 1992, Durham, NC.
4. Moon RE. Treatment of gas bubble disease. Probl Respir Care 1991;4(2):232-252.
5. Myers RA. National registry at MIEMSS 1967-1992. Baltimore, Md: University of Maryland, Maryland Institute for Emergency Medical Services Systems.
6. Nichols G. History of diving. Alert Diver May-June 1993: 4-7.
7. Thom SR. Hyperbaric oxygen therapy: A committee report. Bethesda, Md: Undersea and Hyperbaric Medical Society, 1992:25-27.